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Alcohol, Addiction, and Public Health

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1. Introduction

Addiction can be defined in various ways and in the past 40 years, there have been notable changes in the medical definition of substance dependence, the main medical category to capture addiction (Room, 1998). The current ICD-10 definition (World Health Organization, 1993b; World Health Organization, 1993a) comprises at least three of the following criteria: strong desire or compulsion to use; impaired capacity to control use in terms of onset, termination or levels of use; physiological withdrawal state when substance use has ceased or been reduced, or use to relieve or avoid withdrawal; tolerance (i.e. increased doses are required in order to achieve effects originally produced by lower doses); progressive neglect of alternative pleasures or interests; and persisting with use despite harm. The DSM-IV definition for dependence (American Psychiatric Association, 1994) is quite close (Rounsaville, Bryant, Babor, Kranzler, & Kadden, 1993).[4] However, the medical definition reflects only one view of addiction, mainly from a clinical perspective.

Other views of addiction have been offered (West, 2001; and the other contributions in the January edition of ***Addiction*** 2001) and this contribution will try to sketch out current evidence on alcohol and addiction from three different perspectives, which are only in part overlapping with the medical definition:

- biology and neuroscience
- psychology
- behavioural economics.

We will not only provide evidence in what way alcohol can be considered as an addictive drug, but will also provide comparative data on addictive properties of different substances, i.e. tobacco and illicit drugs. In addition, the usefulness of the concept of "addiction" for alcohol policy and public health will be discussed.

[4] The high agreement is true for the definitions of dependence but less so for ICD-10 harmful use and DSM-IV alcohol abuse (Rounsaville et al., 1993).

In addition, we will examine, how the different theories are able to explain the experiential basis of alcohol addiction, which can probably be best characterised by a gradual change from recreational use to more intoxication and finally addictive use, subjectively defined as successively losing control (see autobiographical literature, (Heyman, 1996); or the life-stories from Alcoholics Anonymous[5], (Mäkelä et al., 1996)). Often there are phases and attempts to stop and reduce alcohol use, to regain control, followed by periods of uncontrolled heavy use, until alcohol becomes the dominant feature of organizing daily life. Finally, some dramatic event ("hitting bottom") marks the change, when the alcoholic regains control over his life.

2. Epidemiology of alcohol use and addiction

In the first section, we will give an epidemiological overview on the prevalence of alcohol addiction. Table 1 summarizes average volume of consumption and prevalence of dependence for different regions of the world (Rehm & Eschmann, 2002; Rehm et al., 2003a; Rehm et al., 2003c)

Table 1: Characteristics of alcohol consumption in different regions of the world (population weighted averages)

WHO Region (See definitions below)	Predominant beverage type	Total con-sumption[1]	% drinkers among males	% drinkers among females	Consumption per drinker[2]	% alcohol dependent[3]
Africa D (e.g. Nigeria, Algeria)	Mainly fermented beverages	4.9	47.0	27.0	13.3	0.7
Africa E (e.g. Ethiopia, South Africa)	Mainly other fermented beverages and beer	7.1	55.0	30.0	16.6	1.6
Americas A (Canada, Cuba, US)	> 50% beer, about 25% spirits	9.3	73.0	58.0	14.3	5.1
Americas B (e.g. Brazil, Mexico)	Beer, followed by spirits	9.0	75.0	53.0	14.1	3.5
Americas D (e.g. Bolivia, Peru)	Spirits, followed by beer	5.1	74.0	60.0	7.6	3.2
E. Mediterranean B	Spirits and beer	1.3	18.0	4.0	11.0	0.0

[5] It should be noted, that the accounts of AA members are quite influenced by pressures to construct a "typical" life story and do not necessarily reflect own experiences (Mäkelä et al., 1996; Arminen & Perala, 2002).

(e.g. Iran, Saudi Arabia)						
E. Mediterranean D (e.g., Afghanistan, Pakistan)	Spirits and beer	0.6	17.0	1.0	6.0	0.0
Europe A (e.g. Germany, France, UK)	Wine and beer	12.9	90.0	81.0	15.1	3.4
Europe B 1 (e.g. Bulgaria, Poland, Turkey)	Spirits	9.3	77	57	14.3	0.8
Europe B 2 (e.g. Armenia, Azerbaijan, Tajikistan)	Spirits and wine	4.3	54	33	9.9	0.2
Europe C (e.g. Russian Federation, Ukraine)	Spirits	13.9	89.0	81.0	16.5	4.8
South-East Asia B (e.g. Indonesia, Thailand)	Spirits	3.1	35.0	9.0	13.7	0.4
South-East Asia D (e.g. Bangladesh, India)	Spirits	2.0	26.0	4.0	12.9	0.8
Western Pacific A (e.g. Australia, Japan)	Beer and spirits	8.5	87	77.0	10.4	2.1
Western Pacific B (e.g. China, Philippines, Viet Nam)	Spirits	5.0	84.0	30.0	8.8	0.9

1 Estimated alcohol consumption per resident aged 15 and older, both recorded and unrecorded, in litres pure alcohol per year

2 Estimated total alcohol consumption (in litres of pure alcohol/year) per adult drinker.

3 Estimated rate of alcohol dependence, among those aged 15+.

Source: Based on estimates (Rehm et al., 2001; Rehm et al., 2002; Rehm et al., 2003a; Rehm et al., 2003c) from the WHO Comparative Risk Analysis within the Global Burden of Disease 2000 Study (Babor et al., 2003; World Health Organization, 2002a; World Health Organization, 2002b). Recorded consumption is derived from official or industry figures; unrecorded consumption is estimated from a variety of sources. The percentages of drinkers (drinking at all in the last 12 months) among males and females are derived from population surveys, where possible. Where figures for a country were otherwise unavailable, they were extrapolated from nearby countries on the basis of similarity of alcohol culture. Estimates of the total population of alcohol dependent people are derived from population surveys, especially the World Mental Health Survey (World Health Organization, 2001; World Health Organization, 2002a).

The 15 regional groupings below (which comprise the 191 WHO Member States) below have been defined by WHO on the basis of levels of adult and of infant mortality. A stands for very low child and very low adult mortality, B for low child and low adult mortality, C for low child and high adult mortality, D for high child and high adult mortality, and E for very high child and very high adult mortality (World Health Organization, 2000). WHO's EUR B has been subdivided to separate out the relatively low-consumption southern republics of the former Soviet Union.

Africa	**D**	Algeria, Angola, Benin, Burkina Faso, Cameroon, Cape Verde, Chad, Comoros, Equatorial Guinea, Gabon, Gambia, Ghana, Guinea, Guinea-Bissau, Liberia, Madagascar, Mali, Mauritania, Mauritius, Niger, Nigeria, Sao Tome and Principe, Senegal, Seychelles, Sierra Leone, Togo
Africa	**E**	Botswana, Burundi, Central African Republic, Congo, Côte d'Ivoire, Democratic Republic of the Congo, Eritrea, Ethiopia, Kenya, Lesotho, Malawi, Mozambique, Namibia, Rwanda, South Africa, Swaziland, Uganda, United

		Republic of Tanzania, Zambia, Zimbabwe
America	**A**	Canada, Cuba, United States of America
America	**B**	Antigua and Barbuda, Argentina, Bahamas, Barbados, Belize, Brazil, Chile, Colombia, Costa Rica, Dominica, Dominican Republic, El Salvador, Grenada, Guyana, Honduras, Jamaica, Mexico, Panama, Paraguay, Saint Kitts and Nevis, Saint Lucia, Saint Vincent and the Grenadines, Suriname, Trinidad and Tobago, Uruguay, Venezuela
America	**D**	Bolivia, Ecuador, Guatemala, Haiti, Nicaragua, Peru
Eastern Mediter-ranean	**B**	Bahrain, Cyprus, Iran (Islamic Republic of), Jordan, Kuwait, Lebanon, Libyan Arab Jamahiriya, Oman, Qatar, Saudi Arabia, Syrian Arab Republic, Tunisia, United Arab Emirates
Easter Mediter-ranean	**D**	Afghanistan, Djibouti, Egypt, Iraq, Morocco, Pakistan, Somalia, Sudan, Yemen
Europe	**A**	Andorra, Austria, Belgium, Croatia, Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland, United Kingdom
Europe	**B 1**	Albania, Bosnia and Herzegovina, Bulgaria, Georgia, Poland, Romania, Slovakia, The Former Yugoslav Republic Of Macedonia, Turkey, Yugoslavia
Europe	**B 2**	Armenia, Azerbaijan, Kyrgyzstan, Tajikistan, Turkmenistan, Uzbekistan
Europe	**C**	Belarus, Estonia, Hungary, Kazakhstan, Latvia, Lithuania, Republic of Moldova, Russian Federation, Ukraine
South-east Asia	**B**	Indonesia, Sri Lanka, Thailand
South-east Asia	**D**	Bangladesh, Bhutan, Democratic People's Republic of Korea, India, Maldives, Myanmar, Nepal
Western Pacific	**A**	Australia, Brunei Darussalam, Japan, New Zealand, Singapore
Western Pacific	**B**	Cambodia, China, Cook Islands, Fiji, Kiribati, Lao People's Democratic Republic, Malaysia, Marshall Islands, Micronesia (Federated States of), Mongolia, Nauru, Niue, Palau, Papua New Guinea, Philippines, Republic of Korea, Samoa, Solomon Islands, Tonga, Tuvalu, Vanuatu, Viet Nam

Clearly, average volume and alcohol consumption and dependence vary a lot between regions and are closely related (Pearson correlation of about 0.79). Both were highest in established market economies in Western Europe and the Former Socialist Economies in the Eastern part (Eur A, Eur C; see legend to Table 1 for a complete listing of countries in different WHO regions) and North America (Amr A), and lowest in the Eastern Mediterranean Region (Emr B, Emr D) and part of South East Asia (Sear D).

Average volume of alcohol consumption and alcohol-related harm are less closely related, however, with a Pearson correlation of 0.58. Similarly, the relationship between alcohol dependence and harm is not that close (0.57). Table 2 gives an overview of the composition of alcohol-related disease burden (Rehm et al., 2001; Rehm et al., 2003d; Rehm et al., 2003c; Rehm et al., 2003b).

Table 2: Alcohol related harm in different regions of the world (population weighted averages)

	Developing Countries				Developed countries						World	
	very high or high mortality		low mortality		very low mortality *net calculation*		very low mortality *only burden*		Former Socialist: low mortality			
	AFR-D, AFR-E, AMR-D, EMR-D, SEAR-D		AMR-B, EMR-B, SEAR-B, WPR-B		Amr A, Eur A, Wpr A		Amr A, Eur A, Wpr A		Eur B, C			
	DALYs	%	DALYs	%	DALYs	%	DALYs	%	DALYs	%	DALYs	%
Alcohol dependence and harmful use	2906	22.1	9366	36.7	5100	64.4	5100	53.1	2299	19.6	19671	33.7
Unintentional injuries	5033	38.2	5961	23.4	1571	19.9	1571	16.4	3929	33.5	16494	28.3
Intentional injuries	1689	12.8	2940	11.5	558	7.1	558	5.8	1874	16.0	7061	12.1
Total alcohol related burden in DALYs	13165	100.0	25519	100.0	7897	100.0	9597	100.0	11742	100.0	58323	100.0
Total burden of disease in DALYs	822718		409688		115853		115853		96911		1445169	
% of total disease burden which is alcohol related	1.6		6.2		6.8		8.3		12.1		4.0	

Alcohol dependence contributed between 22% and 64% to total alcohol-related disease burden, based on the region (see Table 2). The impact of alcohol dependence was lowest in developing regions with high or very high mortality, where volume of drinking and prevalence of dependence was relatively low (e.g. Africa and India). In these regions, harm is caused mainly by drinking to intoxication and subsequent injuries. It should be noted that there is reason to believe that social harm is also related to a considerable degree to intoxication in addition to the effects of dependence (Graham, 2003; Babor et al., 2003).

Level of dependence and its impact on burden of disease is highest in established market economies with a pattern of regular and high frequency drinking (Western Europe, North America, Australia, Japan). In these countries, there is enough regular light to moderate drinking, which has been found to have beneficial effects on coronary heart disease (Rehm, Sempos, & Trevisan, 2003e; Rehm et al., 2003c; Rehm et al., 2003b). In other parts of the world with different drinking patterns alcohol is related detrimentally to coronary heart

disease and cardiovascular disease in general (Rehm et al., 2003e; McKee & Britton, 1998). The detrimental effect is most pronounced in Former Socialist Countries like Russia, where high average volume is coupled with detrimental drinking patterns, i.e. frequent irregular heaving drinking occasions outside meals.

Summary on epidemiology

Average volume of alcohol consumption and prevalence of dependence are highly correlated and vary considerably between different regions of the world. Alcohol related harm is related to either average volume of alcohol consumption or alcohol dependence to a lower degree, and additionally determined by other factors such as drinking patterns.[6] Thus, from an epidemiological point of view, alcohol dependence is not a key indicator for alcohol-related harm.

3. Alcohol, the brain and behaviour

There is ample evidence from biology and neuroscience, that alcohol is an addictive substance, as mainly defined by withdrawal, tolerance and reinforcement. The following overview is in part based on the 10th Special Report to the US Congress on Alcohol and Health, which recently summarized some of this evidence (US Department of Health and Human Services, 2000).

[6] A simplified conceptual model would see three processes linking consumption to harm (Rehm et al., 2003b):

- Dependence
- Intoxication
- Biological processes independent of either dependence or intoxication, either beneficial or detrimental. Accepted beneficial effects include the influence of moderate drinking on coronary heart disease, via reduction of plaque deposits in arteries, protection against blood clot formation and promotion of blood clot dissolution (Zakhari, 1997). Examples of harmful effects include increasing the risk for high blood pressure, direct toxic effects on acinar cells triggering pancreatic damage (Apte, Wilson, & Korsten, 1997) or hormonal disturbances (Emanuele & Emanuele, 1997).

Depressant effects of alcohol

Alcohol reduces the pace of brain activity by the mechanisms listed below (US Department of Health and Human Services, 2000). The depressant effects are one of the reasons why alcohol is taken as a relaxant to reduce stress:

- Decreasing the excitatory action of the neurotransmitter glutamate
- Increasing the inhibitory actions of the neurotransmitter GABA

Alcohol related acute withdrawal syndrome

Following the abrupt cessation of heavy drinking, compensatory mechanisms attempting to overcome alcohol's depressing effect on glutamate systems have the effect of increasing glutamate function. This, together with a corresponding down regulation of inhibitory GABA function (see above at depressant effects), causes the brain hyper-excitability characteristic of acute withdrawal syndrome (US Department of Health and Human Services, 2000). The acute withdrawal syndrome is associated with the body's stress response and can cause damage in different parts of the brain (Adinoff & et al, 1998; Becker, 1998).

Neuroadaptation and tolerance

Repeated exposure to a drug produces long-term changes in nervous system, which can explain tolerance. Tolerance refers to compensatory reactions to enable normal functioning in the presence of a drug, that oppose the acute effects of the drug, resulting in a diminished effect of a given dose of the drug over repeated exposures.

The transition from voluntary use to addictive use occurs through a combination of processes, including reinforcement and neuroadaptation (i.e. compensatory adjustments whereby the brain attempts to continue normal functioning despite the presence of alcohol) that result from repeated exposure to the addictive experience. Sensitization, defined as an increased response to a drug effect following repeated uses, may be one of the mechanisms behind the compulsion to engage in the addictive behaviour (Robinson & Berridge, 1993).

Neurobiological view of addiction and relevance to public health

Thus, from a biological perspective, the transition from a voluntary decision to experiment with alcohol to addictive experience is facilitated by neuroadaptation that also result in what is usually called craving, i.e. a compulsive attraction to the addictive experience (see point on craving below). Neurobiological reactions have been found to underlie both the short-term [acute] response to alcohol ingestion, and the establishment of the long-term [chronic] craving (more to craving see below) that characterizes addiction. Some neuroadaptations may be irreversible.

However, even though there is a biological basis, one must stress that these processes do not imply that chronic use or even chronic heavy use will inevitably lead to addiction (see e.g. already the arguments of (Peele & Alexander, 1985)).[7] For instance, Anthony and colleagues (Anthony, Warner, & Kessler, 1994) found in the US-representative National Co-morbidity Survey, that 21% of the male and 9% of the female users developed dependence as defined by DSM. The probabilistic nature of becoming dependent even after continuous heavy drinking is usually explained by concepts of different genetic vulnerability or individual susceptibility (Pickens, Elmer, LaBuda, & Uhl, 1996). (Buck & Finn, 2000) In addition, brain mechanisms often have also been over-simplified (Hyman, 1996) (Littleton, 2001)

While there is a clear biological basis for addictive processes of alcohol, and some knowledge on who will be susceptible, the relevance of this knowledge to reduce alcohol-related burden is not clear, as burden is additionally shaped by an interaction of biological and social factors (Berkman & Kawachi, 2000). In addition, the biological bases for mechanisms like tolerance or withdrawal also does not necessarily imply that these they are

[7] Similarly, craving has shown rather limited power in predicting actual relapse (Drummond et al., 2000; Shiffman, 2000; Shiffman, 2000).

central for the experience of addiction, or for interventions (see also overview, (Stockwell, 1994)).

Comparisons of neurobiological aspects between substances

There has been a flurry of comparative listing on the addictiveness of substances. For the purposes of this overview we will just refer to one of the first of such ratings by J.E. Henningfield and N.L. Benowitz (Hilts, 1994). They ranked six substances (alcohol, caffeine, cocaine, heroin, marihuana and tobacco) with respect to several criteria and we will report the biologically relevant ones below:

- **Withdrawal**: alcohol ranks most serious by both experts, i.e. producing highest level of withdrawal of all 6 substances
- **Tolerance**: alcohol ranks in middle of substances; heroin ranks quite high
- **Reinforcement**:[8] alcohol ranks in middle of substances, after cocaine and heroin

Summary on alcohol, the brain and behaviour

There is a clear biological basis to explain key processes of dependence such as withdrawal and tolerance. Overall, the addictive properties of alcohol can be ranked highest of all substances with regards to withdrawal and in the middle of psychoactive substances for tolerance and reinstatement. While there is a clear biological basis for these mechanisms, the specific relevance of withdrawal and tolerance for public health and alcohol policy seems to be shaped by social factors.

[8] Reinforcement: A measure of the substance's ability, in human and animal tests, to get users to take it again and again, and in preference to other substances.

4. Psychological theories of alcohol and empirical results

Social learning and expectancy theories

Social learning (Bandura, 1977; Bandura, 1994) and specifically expectancy theories have been used both to explain addictive behaviour and to shape interventions such as prevention and therapy. Alcohol expectancies can be defined as structures in long-term memory that have impact on cognitive processes governing current and future consumption. According to social learning theory, the particular alcohol expectancies held by an individual are the result of their direct and indirect past experience with alcohol and the associated environment. Expectancies are usually operationalized by questionnaires comprising a number of positive (example: “I expect to be the life and soul of the party, if I have a few drinks”) and negative expectancies (example: “I expect to have a hangover if I have a few drinks”). Expectancies are considered to be part of the motivation to drink or to refrain from alcohol consumption (Cox & Klinger, 1988; Lang & Michalec, 1990; Jones & McMahon, 1998).

If these theories are correct, positive associations should be found between positive expectancies and consumption and negative associations between negative expectancies and consumption. Associations do not entail cause and this difficulty is especially relevant in a social learning framework in which the potential for reciprocal causality is a key feature (Bandura, 1977).

Jones and colleagues (Jones, Corbin, & Fromme, 2001) recently reviewed evaluations of expectancy theory. They found consistent associations between expectancies and quantity of drinking, with expectancies being more strongly associated with quantity than with frequency of drinking. This finding has been robust among adolescents (Chen, Grube, & Madden, 1994; Fromme & D'Amico, 2000), college students (Mooney, Fromme, Kivlahan, & Marlatt, 1987) (Carey, 1995) and community samples (McMahon, Jones, & O'Donnell, 1994; Lee, Greely, & Oei, 1999). The most important contribution of alcohol expectancies to the

field may be their ability to predict changes in drinking and the development of alcohol-related problems and alcohol dependence symptoms (Jones et al., 2001). However, the magnitude of effects for expectancies on drinking behaviour seems to be small.

Recent reviews of empirical evidence for manipulating expectancies to manipulate consumption have yielded only mixed support (Jones et al., 2001). Thus, while there is overall adequate support for the empirical association of alcohol expectancies and consumption, the transfer into intervention techniques has not been as successful as hoped.

Craving

Craving is often described as a strong need, urge or compulsion, to drink, and thus has been included in the definition of alcohol dependence (see above). In the end 80s and 90s, the concept of craving was revered as unifying concept to subsume biological as well as behavioural aspects of dependence and serve as a theoretical guide to develop better interventions, both behavioural and pharmaceutical (see the NIAAA workshop in 1997 on "Treatment and Alcohol Craving: Expanding the Paradigm"). However, despite calls to tighten the use of the term craving (Kozlowski & Wilkinson, 1997), the terms has still many and different meanings to different disciplines, and consequently different operationalizations (Drummond, Litten, Lowman, & Hunt, 2000; Shiffman, 2000). Thus, to give just one example, it is not clear if the results of neuroimaging techniques used by neuroscientists capture the same phenomenon that patients experience and verbalize as urge or "craving". Similarly, the link between animal models and human models of craving must be made more explicit in order to judge on the usefulness of the craving concept as a key construct for addiction (Li, 2000).

Current research summaries tend to be cautious about the usefulness of craving to explain dependence and addiction (Drummond et al., 2000; Drummond, 2001), as many prospective clinical studies have concluded that craving does not reliably predict relapse and that the

concept is of little or no clinical utility. Contrary to earlier more simplistic clinical models of addiction, more recent models do not require that craving be present for relapse to occur. Thus, new approaches to study human craving have been created, which may enhance its predictive validity and yield more knowledge of its nature, course, behavioural sequelae and regulatory function in alcohol/drug consumption.

Summary on psychological theories of alcohol and empirical results

Overall, there are clear associations between alcohol expectancies and drinking behaviours. However, the causal direction is not always clear and the transfer of expectancy concepts into interventions has seen mixed results. The concept of craving has been conceptualised in different theoretical models but overall, while there is promise, there is no “craving theory” which is empirically supported in all key areas of research including biological research.

There is some doubt about the overall explanatory and predictive power of psychological constructs for addictive behaviour. Many theories seem to be descriptions or epiphenomena of behaviour rather than independent constructs, which can explain subsequent behaviour.

5. Behavioural economics of alcohol use and addiction

Behavioural economics theories

Since addiction is a behaviour which seemingly contradicts basic economic rational choice assumptions,[9] it has attracted lots of theorizing, and thus, there are theories which are intended to explain addictive behaviour (Becker & Murphy, 1988; Rachlin, 1997; Heyman, 1996; Elster & Skog, 1999; Vuchinich & Heather, 2003b). All of these theories owe to the original behavioural theory of choice, the matching law (Herrnstein, 1970). This law is different from classic psychological explanations of behavior in that:

a) it related the attractiveness of behavioural alternatives; i.e. it was a relative account of behavior[10]
b) it related aggregates of behavior to aggregates of reinforcement over some extended temporal interval; i.e. it was a molar rather than molecular account of behavior.

Four theoretical behavioural economic theories of addiction can be distinguished:

- hyperbolic discounting (Ainslie, 1992; Ainslie, 2001)
- melioration theory of addiction (Herrnstein & Prelec, 1992; Heyman, 1996)
- the relative theory of addiction (Rachlin, 1997; Rachlin, 2000)
- theory of rational addiction (Becker et al., 1988)

All of these theories have distinctive features, they share the element of time discounting, and conceive the addict as a person who is living in the present, being insufficiently motivated by the future (Skog, 2003). The way into heavy drinking and finally addiction has been described as the “primrose path”, seeking immediate pleasure not being sufficiently motivated by the gradual deterioration that follows from continued heavy consumption (Skog,

[9] Simply speaking the question is why people engage in behaviour such as drinking heavily or becoming addicted to drinking if they know that it can or will have negative consequences. Rationality implies that the person acts according to his or her own consistent preferences and does not yield to wishful thinking. Consequently, assuming that people judge the consequences of heavy drinking as negative, they should not engage in heavy drinking.

[10] The classic paradigm was relating response to stimuli triggering these responses without analysing the general context of other behavior and other consequences.

2003). This basic idea has been empirically supported; for instance Vuchinich and Simpson found that problem drinkers and heavy social drinkers discount the future more heavily than light drinkers (Vuchinich & Simpson, 1998). However, overall, there seem to be more theories than empirical data especially when it come to testing different, partly overlapping and partly discordant theoretical assumptions against each other (Vuchinich et al., 2003b). In addition, it seems that much of the theories are driven by theoretical considerations and the motivation to prove that seemingly irrational behaviour can in fact be brought into congruence with rational theories.[11]

Taxation or price changes and consequences for consumption and harm

The effect of price changes on alcohol consumption and alcohol-related harm has been more extensively investigated than any other potential alcohol control measure. Econometric methods have been the most common tool used to study these effects. Combining the studies referenced in different reviews, econometric data on the relation of price to alcoholic beverages or certain categories of alcoholic beverages are currently available at least from the following countries (Babor et al., 2003): Australia, Belgium, Canada, Denmark, Germany, Finland, France, Ireland, Italy, Kenya, the Netherlands, New Zealand, Norway, Poland, Portugal, Spain, Sweden, the United Kingdom, and the United States (Huitfeldt & Jorner, 1972) (Lau, 1975) (Ornstein, 1980) (Ornstein & Levy, 1983) (Godfrey, 1986) (Olsson, 1991) (Clements & Selvanathan, 1991) (Yen, 1994) (Edwards et al., 1994) (Österberg, 1995) (Österberg, 2000). This list indicates that information about the effects of changing alcohol prices on alcohol consumption chiefly derives from the developed countries.

[11] It may also be, that the preferences of people may be misperceived by conventional norms. Orford and colleagues (Orford et al., 2002) examined 500 heavy drinkers and found that for them, perceived benefits outweighed the drawbacks in both forced-choice ratings and open-ended interviews. Thus, their excessive drinking behaviour could not be considered irrational or being in contrast with their own preferences.

Overall, the price-elasticities[12] for alcoholic beverages estimated in different studies have shown that when other factors remain unchanged, an increase in price has generally led to a decrease in alcohol consumption, and that a decrease in price has usually led to an increase in alcohol consumption. In other words, alcoholic beverages appear to behave in the market like most other consumer goods and in the way presupposed by the theory of consumer demand.

While demonstrating that the use of alcohol is similarly constrained by economic circumstances, studies dealing with different countries and time periods have found different values for price-elasticity with respect to both total alcohol consumption and the consumption of different categories of alcoholic beverages. For instance, in the United States estimated values of price-elasticity for beer range from approximately zero to -1.4, estimates for wine range from -0.4 to -1.8, and estimates for distilled spirits from -0.1 to -2.0 (Österberg, 1995).

In econometric studies based on time series data, the price-elasticity values in many ways reflect the average reactions of consumers to changes in prices. It is particularly the treatment of alcohol consumers as a single homogenous group that has raised concerns about the policy implications of price elasticity estimates. One example of these concerns is the disagreement in the literature on whether heavy drinkers or addicts are responsive to changes in alcohol prices.

Econometric research in the United States, for example, has been extended to the study of the relationships between beverage taxes and self-reported use of alcohol among specific

[12] Economists use the term **price elasticity of demand** when measuring the sensitivity of consumption to changes in price. The price elasticity of demand is defined as the percentage change in consumption resulting from a one percent change in price. For example, a price elasticity for alcohol of –0.5 implies that a 1% increase in price would reduce alcohol consumption by 0.5%. Three types of elasticity are distinguished. If the price elasticity of demand has a value between 0.0 and –1.0, the demand for a commodity is said to be 'inelastic' with respect to its own price, as a change in its price results in a relatively smaller change in its consumption. If the own-price elasticity has a value of –1.0, the demand is said to be **unit price elastic**, as the change in its price results in an equal relative change in its consumption. Finally, with values below –1.0, the demand is said to be elastic, as the change in its price leads to a proportionally greater change in its consumption. Obviously, commodities with very inelastic demand are the best alternatives for the purposes of revenue generation. While alcohol demand is clearly influenced by price, it has more often been found to be inelastic than elastic (see text).

demographic groups, particularly young drinkers (Chaloupka, Grossman, & Saffer, 2002).These studies typically relate some measure of beverage prices or taxes to self-reports of alcohol use obtained from large national surveys. Grossman and colleagues estimated the effects of price on youth alcohol use (Grossman, Coate, & Arluck, 1987; Coate & Grossman, 1988). Their studies concluded that beer consumption by youths was inversely related to both the monetary price and the minimum legal drinking age. In addition, they concluded that frequent or heavy drinkers were more sensitive to price than infrequent or light drinkers (Grossman et al., 1987). Similar research indicated that higher beer excise taxes significantly reduced both the frequency of youth drinking and the probability of heavy drinking (Laixuthai & Chaloupka, 1993), and that beer prices had a significant effect on underage drinking and binge drinking among female college students (Chaloupka & Wechsler, 1996).

An important issue for policy is the extent to which adult heavy and problematic drinkers or addicts are responsive to changes in alcohol prices. There are mixed results on this question (Manning, Blumberg, & Moulton, 1995; Boys et al., 2002). However, most research has used cross-sectional data, which is weak in terms of causal significance. However, there is experimental literature, showing that changes in prices can affect heavier drinkers and people who are alcohol dependent.

In the early 1970s many bars, taverns, and restaurants in many Western countries initiated a variety of sales promotions, called “happy hour”, to attract more customers. These measures included temporary reductions of alcohol prices. They could take the form of selling two drinks for the price of one or a 25 per cent reduction for all beverages or free beverages for a particular type of patron, or they could give a fixed price for everything one could drink in a certain time interval. Consequently, “happy hour” offers one possibility to study the effect of decreasing prices of alcoholic beverages on drinking among different consumers.

Babor et al. (1978) created an experimental analogue of the “happy hour” situation by either giving up alcohol under a single price condition (50 cents per drink) or a daily price reduction during 3 hours in the afternoon (25 cents per drink). The results demonstrated that the afternoon price reduction increased alcohol consumption during happy hour both for casual and heavy drinkers. When the purchase price was reduced by half, casual and heavy drinkers increased their consumption eight and nine times, respectively, over that of matched controls without happy hours. Although this increase happened during hours of normally light intake, it is important, that drinking during happy hours was not a substitute for consumption during other times of the day. Thus, the additional drinking during happy hour was superimposed on their more typical pattern of consumption over the 20-day period. Reinstatement of the standard purchase price following the happy hour period resulted in a normalization of the daily drinking pattern. An exploration into the generalizability of findings in a more natural condition of a barroom setting confirmed the results: a discount drink policy (“happy hour”) could be related to both increases in frequency of drinking and amount of alcohol consumed (Babor, Mendelson, & Uhly, 1980). In the same tradition of experimental studies, Bigelow and Liebson (1972) could show that varying the costs of drinking was effective in modifying drinking behavior of alcohol dependent subjects. Thus, heavy drinkers including alcohol dependent subjects have been consistently shown to be influenced by price in experimental settings (Mello, McNamee, & Mendelson, 1968).

Summary on behavioural economics of alcohol use and addiction

The most fundamental law of economics links the price of a product to the demand for that product. Accordingly, increases (or decreases) in the price of alcohol (i.e., through tax increases/decreases) would be expected to lower (increase) alcohol consumption and its adverse consequences. Studies investigating such a relationship found that alcohol price was one factor influencing alcohol consumption in the predicted way. Price affected consumption, including consumption of problem drinkers and people with alcohol dependence.

However, there has been also significant variation in consequences following different taxation and/or price changes. Behavioural economics theories may provide insights to explain these variations as well as other key questions in the emergence and maintenance of addictive behaviour. However, in their current state, although there are different competing theories attempting to explain alcohol consumption as rational behaviour, there is not enough empirical evidence to decide about competing theories.

6. Alcohol as addictive substance – Conclusions and consequences for public health and alcohol policy

Alcohol is an addictive substance by current definitions. In fact, compared to other substances, the addictive potential of alcohol is relatively high. Repeated consumption of alcohol leads to changes in the brain, some of which are irreversible. However, what does this mean for public health and alcohol policy?

Neuroadaptation in the brain do not mean, that addicts are unable to choose, nor that they cannot be influenced by the situation and external interventions. Every behaviour has a neurobiological substrate. Many behaviours lead to permanent changes in the brain, i.e. neuroadaptation. This does not mean, that these behaviours are not subject to choice. Skog (2003) gives the example of learning how to ride a bicycle: it is evident that when a person learns to ride a bicycle, there will occur some changes in the cyclist's brain. Some of them will be permanent. However, we would not consider bicycling as a behaviour, which leaves us no choice.

Alcohol addiction may be different to bicycling and it has been argued that it creates an "internal compulsion" that prevents choice. However, the concept of compulsion has not yet been defined in a way that it could combine neurobiology, subjective experiences and displayed behaviour (see point above), as well as the current notion of internal compulsion may be conceptually incoherent (Watson, 1999). So there are good arguments for stating that addicts can chose, even though not all instances of drinking are best understood as results of rational choices (Skog, 2000, 2003; Vuchinich & Heather, 2003a). This point is also supported by earlier experiments of Mello and colleagues (Mello et al., 1968) (Mello & Mendelson, 1972). People with alcohol dependence were experimentally confronted with different situations, including unrestricted access. Results showed that alcoholics did not tend to drink maximum amounts available, did not drink to a state of oblivion, but organized

their time rather systematically including relatively abstinent work periods with partial withdrawal symptoms.

From a public health perspective with regards to alcohol and alcohol policy, the details of the discussion on choice may be irrelevant, however. First of all, alcohol-related harm is often unrelated to addiction. As laid out above, a substantial portion of this harm is related to intoxication or physiological processes triggered by alcohol use *per se* irrespective of the presence or absence of addiction.

The price of alcohol also determines consumption in humans, in contrast to some results from animal studies (Heyman, Gendel, & Goodman, 1999). Thus, there is good evidence, that changes in taxation or other price changes for alcoholic beverages influenced level of consumption (i.e. the higher the price/taxation, the lower the consumption and vice versa). For the discussion here it is irrelevant, if the influence of price on people with alcohol dependence or problem drinkers was smaller than on light drinkers. The important fact for public health is that there was an influence on all drinkers. Moreover, there is an emerging literature linking alcohol-related harm (e.g. liver cirrhosis, motor vehicle collisions, domestic violence, homicides) directly to taxation and prices (Babor et al., 2003).

Alcohol taxes are thus an attractive mean to politicians as they can be used to both, generate direct revenue because of the relative inelasticity in demand, as well as reduce social costs because alcohol-related harms will be reduced. In achieving this goal the most important aspect seems to be that of potential alternatives or substitutions to taxed alcoholic beverages: both in terms of illegal smuggling or illegal production within the country. These aspects have to be carefully balanced, as well as all aspects of differential taxation of different kinds of beverages. Special care should be given to preferred beverages of heavy users, both in terms of heavy drinking occasions and in terms of heavy average volume.

But taxation is only one of several environmental changes that have been empirically shown to reduce alcohol-related harm. Changes in availability or random breath testing would be other examples for such interventions. What is necessary at this point are comparisons on effectiveness and cost-effectiveness of these interventions in different environments, including data on combined interventions. The concept of addiction is not very helpful here, as from a policy point of view, all alcohol-related harm should be considered and addiction cannot even serve as an empirical indicator for this main outcome of interest. Thus, from the perspective of public health or alcohol policy, we conclude that addiction is not a useful concept. Rober MacCoun (MacCoun, 2003) comes to a similar conclusion with respect to the usefulness of addiction to policy in the area of illegal drugs.

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